Costs of Health Damage from Atmospheric Emissions of Toxic Metals. Part 1: Methods and Results

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Abstract

Significant quantities of toxic metals are emitted to the air by the incineration of waste, as well as by the combustion of coal and oil. To optimize the regulations for their emissions one needs to know the cost of their damage. That requires an impact pathway analysis, with realistic dispersion models, exposure-response functions and monetary values. In this paper we explain the method and assumptions and present results for arsenic, cadmium, mercury and lead, the most important toxic metals in terms of damage cost. We also estimate their contribution to the damage cost of waste incineration and electric power from coal for typical situations in Europe. The damage costs of As, Cd, and Pb are much higher than previous estimates, because of a large number of new epidemiological studies, implying more and more serious health effects than what had been known before. New cost-benefit studies for the abatement of toxic metal emissions are advisable. The discussion of the epidemiological studies and the derivation of exposure-response functions are presented in two companion papers, one for As and Cd, the other for Hg and Pb.

Key words: toxic metals, arsenic, cadmium, lead, mercury, damage cost, external cost, exposure-response functions

1. Introduction

Toxic metals are emitted to the air by several industrial sources, especially the incineration of waste and the combustion of coal and oil. Even though stringent new regulations for waste management in the EU and elsewhere have brought about impressive improvements, the remaining emissions are still significant in terms of damage costs, as this series of papers shows.

To optimize the regulations for their emissions one needs to know both the cost of their damage, also known as external costs, and the cost of actions to reduce the emissions. Here

we provide estimates of the damage cost per kg of emitted pollutant, for typical conditions in Europe. Because of the length of the documentation and calculations we split the presentation in three parts. In the first we explain the method and assumptions and summarize the results; as illustration we also look at implications for waste incineration and for electricity production from coal. Detailed derivation of the results can be found in Parts 2 and 3, respectively for Hg and Pb, and for As and Cd [1].

The focus on these metals has been motivated by the prioritization study of Nedellec, Lapkoff and Rabl [2012], who ranked Arsenic (As), mercury (Hg), cadmium (Cd), nickel (Ni), manganese (Mn), chromium (Cr-VI) and lead (Pb) among the 12 most worrisome pollutants emitted by waste treatment facilities, taking into account both emitted quantities and general toxicological criteria. Whereas for As, Cd, Hg and Pb there are sufficient epidemiological studies, for Cr-VI and Ni only toxicological studies are available for the most worrisome health endpoints and the methodology is a bit different, to be addressed in another publication [2].

The calculation of damage costs requires an impact pathway analysis (IPA), with realistic dispersion models, using exposure-response functions (ERF) [3] and monetary values for each of the possible health endpoints. The principal steps of an IPA can be grouped as follows:

- Emission: specification of the pollution source (e. g. emission of As by an incinerator with such and such stack height at such and such location);
- Dispersion: calculation of increased pollutant exposure in all affected regions;
- Impact: calculation of the impacts (damage in physical units) due to the increased exposure, using ERFs (e. g. cases of cancer due to ingestion of As);
- Cost: monetary valuation of these impacts (e. g. multiplication by the cost of a case of cancer).

The methodology for IPA has been developed by the ExternE project series in the EU [ExternE 2005, 2008] and by analogous projects by EPA [Abt 2004] and National Research Council in the USA [NRC 2010]. For a general review and summary of results see Rabl, Spadaro and Holland [2014]. The focus of these projects has been on greenhouse gases and the classical air pollutants (PM, NO_x, SO_2, etc) that are emitted in large quantities; the toxic metals have not yet received sufficient attention.

Another method, LCA (life cycle assessment), frequently employed for environmental analysis, is not suitable because it does not use realistic ERFs for toxic metals and excludes monetary valuation. We also note that the rationale for damage costs is very different from the current practice of much risk assessment which uses the upper bound of the 95% confidence interval for the health end point with the lowest threshold, in order to determine exposure limits that entail negligible risk. Damage costs have to be calculated as expected values, summed over all end points, rather than as upper limits.

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[1] Part 2 is being published in the same issue, Part 3 is not yet published but available at www.arirabl.org/software/.

[2] We also estimated a damage cost for Mn (not a large number per kg) but hesitate to publish it because of questions about the chemical form of exposures from industrial emissions in view of the fact that Mn is an essential element for the body.

[3] The term dose-response function is also widely used, but we prefer ERF because exposure is a more general term that can refer equally well to ambient concentrations and to intake or absorbed dose.

Here we focus on health impacts because the damage cost assessments in the USA and Europe have found that the health impacts of pollutants (other than greenhouse gases) impose much higher costs than other impacts such as ecosystem impacts – a finding we find very plausible as explained at the end of Section 3.

In recent years much progress has been made with regard to the epidemiology of toxic metals. In the past most studies of toxic metals were based on relatively small cohorts of industrial workers whose exposures were extremely uncertain, with results that may not be representative of the general population because industries tend to recruit workers in better than average shape. Now many studies of the general population have been carried out. The measuring of exposures has been improved by using dietary surveys and biomarkers. Numerous national biomonitoring surveys, such as NHANES (National Health and Nutrition Examination Survey), provide detailed data of biomarkers of pollutants for thousands of individuals. Here we use such data for determining the fraction of the incremental collective exposure that is above threshold.

An analysis of damage costs involves numerous choices for the interpretation of studies and the selection of parameters. Whereas readers may well disagree with our choices, we offer a framework where it is easy to modify the parameters for alternative calculations, and we have made the spreadsheet with the calculations available as Supplemental file at the Risk Analysis site [4].

2. Material and Methods

2.1. Intake fractions

The damage depends on the site of the emitting source (especially the population density of the surrounding population, but also the meteorological and geographic conditions) and the height of the source. This variation of the damage can be very strong for the inhalation impacts of primary air pollutants: for example PM from cars in large cities has a damage cost about two orders of magnitude higher than the same PM if emitted from tall stacks in rural zones. But for power plants and incinerators the variation with site is much smaller (less than a factor of about three) because they have tall stacks (which reduces the importance of the local impacts) and they tend to be in zones with relatively low population densities.

Furthermore, for atmospheric emission of toxic metals the dose due to ingestion is about two orders of magnitude larger than the inhalation dose [5]. Since the ingestion dose involves the production and transport of food over hundreds of km, the variation with emission site is reduced so much that one can calculate the exposure based on regional average values of population density, food intake and environmental parameters (such as meteorology, soil characteristics and types of agricultural production).

It is convenient to use intake fractions (IF). IF is defined as the fraction of the mass of an emitted pollutant that will pass through a human body. Here we use the IFs of Spadaro and Rabl [2004] for typical conditions in Europe, listed in Table 1. Their calculation is based on the multimedia model of EPA [1998] and can be downloaded from www.arirabl.org. For

[4] It is also available at www.arirabl.org/software/. Due to rounding some numbers in the tables may not appear entirely consistent; the calculations are done with full precision.
[5] For an explanation see Section 8.4.6 of Rabl, Spadaro and Holland [2014].

other regions the IF have to be scaled in proportion to the regional population density (within a radius of about one thousand km). These IF are the collective dose in mg per emitted kg, without regard to the fraction that is actually absorbed by the body. They are based on an average respiration rate of 20.6 m^3/day, a water intake of 0.6 m^3/yr and a typical European diet; for other diets the numbers in Table 1 can be adjusted in proportion to the respective food sources. The IF from water should be reduced to the extent that water treatment plants filter out part of the metals. In Table 1 we do not show Hg because it requires a very different pathway analysis, with dispersion over the entire hemisphere, by far the most serious impacts being due to methyl-Hg in fish, see our companion paper on Hg and Pb.

Table 1. Intake fractions (IF) for typical conditions in Europe, in mg per emitted kg. From Spadaro and Rabl [2004].

Pathway	**As**	**Cd**	**Cr VI**	**Ni**	**Pb** [a]
Inhalation	3.9	3.9	3.9	3.9	3.9
Water	31.1	31.7	31.0	31.5	35.5
Cattle milk	156.2	0.3	38.0	27.2	10.8
Cattle meat	13.8	1.4	36.8	43.9	4.1
Freshwater fish	15.6	31.7	1.6	31.6	9.0
Grains	60.8	119.5	60.4	64.4	80.4
Root vegetables	12.4	24.1	12.0	13.1	16.0
Green vegetables	16.2	47.5	15.9	17.7	24.0
TOTAL	**310**	**260**	**200**	**233**	**184**

[a] values for Pb have been recalculated assuming the same deposition velocity of PM_{10} as for the other metals (instead of the one for $PM_{2.5}$ in the original paper).

Ideally one would of course prefer to have the relevant site-specific results for each policy choice. However, in practice they are unlikely to be available because the multimedia calculations needed for damage costs of toxic metals are too complex. It is therefore reassuring to know that for metals the variation with site is sufficiently small to allow the use of typical damage costs. Specifically the sensitivity analysis of Spadaro and Rabl [2004] and comparison with a site-specific model [Bachmann et al 2008] indicate that site variation within a region is unlikely to be more than a factor of two, not significant in view of the overall uncertainties (see Section 2.4).

2.2. Exposure-response functions (ERF)

The ERF is a central ingredient in the assessment of health impacts. The regulatory approach developed by agencies such as EPA and WHO distinguishes cancers due to genotoxic carcinogens from other endpoints. Whereas the ERFs for genotoxic carcinogens are assumed to be linear without threshold, for other endpoints only thresholds are considered in order to answer the question whether or not there is a significant risk for the population (i.e. whether the exposure is above or below the threshold) [6]. As discussed in a report by the National Research Council [NRC 2009], such dichotomy poses problems because thresholds are not sufficient for quantifying the impacts if there is exposure above the threshold. That report

[6] But the practice is evolving, for instance the EU's REACH Regulation goes beyond only considering thresholds for non-genotoxic endpoints.

recommends developing explicit ERFs for non-cancer endpoints. That is what we do for toxic metals in the present series of papers.

To identify the relevant ERFs we have carried out an extensive literature review, beginning with general toxicological profiles by World Health Organization (WHO), EPA and Agency for Toxic Substances and Disease Registry (ATSDR). Then we searched Medline PubMed with the criteria:

(effect OR effects) AND health AND "name of metal",

going back to 1990. We decided to use mainly epidemiological studies for the determination of ERFs, relying on toxicological studies for supplemental information about mechanisms of action and the question of thresholds. The reason for this choice is that for the most important impacts there is now a sufficient body of epidemiological studies and they allow a more reliable impact assessment than the extrapolation of toxicological studies to humans.

In selecting the studies for deriving an ERF, we give preference to: prospective cohort studies, general population studies, individual exposure measurement, and low exposure levels. Rationales for the selected studies are detailed in the companion papers. And of course a key consideration is whether there is sufficient evidence from the totality of the literature for the causality of a specific end point due to a specific metal.

Most epidemiological studies of pollution provide their results in the form of relative risk (RR) for the occurrence of an endpoint as a function of an exposure indicator, relative to a reference level. To construct an ERF one also needs to know the reference incidence rate in the population. We find it convenient to state incidence rates in units of cases per year per average person. Thus we obtain the ERF slope (s_{ERF}) as

$$s_{ERF} = \text{incidence rate} * \Delta RR/\Delta E, \text{ with } \Delta RR = RR - 1 \quad (1)$$

where ΔRR is the RR increase for an increase ΔE of the exposure. Since we state the exposure E as intake rate in units of (mg/yr), the units of s_{ERF} are (cases/yr)/(mg/yr) or simply cases/mg.

Typically the epidemiological studies report the RR for effects observed after exposure during a specific period in time. The exact relation between time and duration of exposure and the resulting effect is difficult to establish: usually the data are insufficient for firm conclusions. For damage cost calculations it is most reasonable to assume stationary conditions, by interpreting the effect rate measured in a study as being due to constant exposure at the measured level. The damage cost is based on lifetime impact due to lifetime exposure. For the calculations in our spreadsheet we find it convenient to prorate exposures and impacts to one year.

For some endpoints additional data are required. For studies that are based on biomarkers (urine or blood concentration of the pollutant or their metabolites) rather than exposure or intake of the pollutant one has to estimate a conversion factor from biomarker to exposure or intake.

For deaths due to specific identifiable causes, such as cancer deaths, it is sufficient to use the corresponding reference mortality rate. But many studies of mortality due to pollution consider a change in all-cause mortality, without identifying specific causes. Typically such studies report RR as change in the age-specific mortality rate $\mu(x)$, defined as the fraction of a cohort between the age x and x + 1 year who have died during the year. As explained by Rabl

[2003] it makes no sense to calculate a corresponding number of deaths for all-cause mortality [7]; only loss of life expectancy (LE) can be calculated unambiguously. For such mortality we use life table data of France to calculate the loss of life expectancy (LE) corresponding to the RR observed in an epidemiological study. The French data for 2005 are representative of most of the population of the EU and have the additional advantage of including detailed annual data for all ages up to age 104, unlike most other databases that stop around age 85. We find the following relation between LE loss and increase in RR, per year of exposure

$$\Delta LE/\Delta RR = 0.148 \text{ YOLL/yr per } \Delta RR, \qquad (2)$$

where YOLL (years of life lost) is used as unit to designate the LE loss. For the calculation of this number we have assumed an increase $\Delta RR = 0.1$ that affects all ages above 10 (the latter chosen to account for a lag between exposure and effect), and we have divided the lifetime change by an effective exposure duration of 70 yr. The number varies somewhat with ΔRR, from 0.141 at $\Delta RR = 0.20$ to 0.151 at $\Delta RR = 0.05$, but Eq.2 is a good compromise because a ΔRR of 0.1 is fairly representative of policy choices.

A crucial and controversial issue is the form of the ERF at low exposures, in particular whether there is a no-effect threshold. The simplest form is a straight line through the origin. Such linearity without threshold is also by far the most convenient for the calculation of damage costs. With linearity the calculations are conceptually clear and numerically simple. It does not matter how the intake fraction or collective dose is distributed among the population and the total damage is equal to the damage of an average person receiving the entire intake fraction.

Linearity without threshold is the appropriate form on theoretical grounds when the effect of a pollutant occurs through a mechanism that is also active without the pollutant. In that case there is, so-to-speak, an underlying ERF associated with that mechanism for which the entire population is above threshold. The effect of an incremental exposure is additive to the background occurrence, and continuity implies proportionality between a small incremental exposure and its effect. Such is the case for genotoxic carcinogens since DNA damage is an ever present background process. In view of the available evidence linearity without threshold is also most plausible for particulate air pollution and for the neurotoxic effects of Hg and Pb. But it is not a general rule, as shown by the "mega-mouse experiment" of Frith, Littlefield and Umholtz [1981].

For the present paper we find it most appropriate to begin with the assumption of linearity without threshold, as an upper bound for the impacts. Then we calculate how the numbers would change in the presence of a threshold, assuming a hockey stick for the form of the ERF (even though in reality population-level ERFs do not have a sharp threshold because of individual differences in sensitivity to pollution). We base the thresholds on limit values recommended by organizations such as EPA for maximum safe exposure.

[7] Multiplying ΔRR $\mu(x)$ by the cohort size seems natural but it is wrong because it does not account for induced changes in the size of age cohorts in subsequent years: everybody dies exactly once, regardless of any change in RR; see Rabl [2003].

2.3. Calculation of physical impacts

With the assumption of stationary conditions one can calculate a constant impact rate ΔI, e.g. the rate of the health endpoint per year, corresponding to a constant emission rate ΔQ, e.g. 1 kg/yr, and a constant collective intake rate ΔE in mg/yr. In the absence of a threshold the impact rate is

$$\Delta I = s_{ERF}\, \Delta E \tag{3}$$

and has units of cases/yr. Dividing the impact rate by the emission rate one obtains the impact per emitted quantity, for instance the cancer deaths per kg. Since the intake fraction IF is the collective intake per kg of emitted pollutant, in units of mg/kg, the intake rate ΔE can be replaced by

$$\Delta E = IF\, \Delta Q\ . \tag{4}$$

Multiplication of the impact rate by the cost P per case for the endpoint in question yields the damage cost rate, in units of €/yr,

$$\text{damage cost rate} = P\, s_{ERF}\, IF\, \Delta Q\ . \tag{5}$$

Dividing by the emission rate we obtain the damage cost D in € per kg as

$$D = P\, s_{ERF}\, IF \tag{6}$$

where

s_{ERF} = ERF slope, in units of cases/mg, and
P = unit cost ("price"), in units of €/case,
IF = intake fraction = collective dose per kg of emitted pollutant, in units of mg/kg.

Because the time base for our calculations is 1 yr, we find it convenient to multiply the customary intake rates by 0.36525 to convert them from μg/day to mg/yr.

Some ERFs are stated in terms of concentrations in ambient air or drinking water rather than intake. In particular, the ERFs of the IRIS website of EPA for cancers due to inhalation (called unit risk factors) are stated as lifetime cancer risk per concentration in the ambient air and based on a lifetime exposure of 70 years. The inhalation unit risk factors can be expressed as equivalent s_{ERF} with units of (cases/mg) for use in Eq.4 with the inhalation IF, if one takes the inhalation rate of 20.6 m^3/day assumed by Spadaro and Rabl [2004] to obtain the inhalation dose in (mg/yr)/(μg/m^3). However, we also note that the unit risk factors are the upper bound of the 95% confidence intervals rather than expected values; therefore one also has to multiply by the ratio central value/upper bound. Thus we obtain the ERF slope in units of (cancers/yr)/(mg/yr), i.e. cases per mg of intake,

$$\begin{aligned} s_{ERF} &= (1/70) * \text{Unit Risk}/(20.6*365.25/1000\ (\text{mg/yr})/(\mu\text{g/m}^3)) \\ &\quad * (\text{central value/upper bound}) \\ &= (\text{Unit Risk/yr}) * 0.0019 * (\text{central value/upper bound})\ (\mu\text{g/m}^3)/(\text{mg/yr}) \end{aligned} \tag{7}$$

where Unit Risk = cancers per lifetime per (μg/m^3). Unfortunately the IRIS websites do not provide any information for the ratios of central value/upper bound. For Parts 1, 2 and 3 of

this series that does not matter because the cancers calculated with the unit risk factors of EPA are negligible compared to what we find from more recent epidemiological studies.

2.4. Monetary valuation

The monetary valuation is based on the willingness-to-pay (WTP) for avoiding a loss or for gaining an improvement; thus it includes not only market costs but most importantly the cost of pain and suffering. We use the unit costs listed in Table 2, as explained in the following (with some rounding of the numbers). All these unit costs are uncertain, but the reader can readily modify the damage costs for different values.

The most important items are for mortality, in particular the value of a prevented fatality (VPF) [8] and the value of a life year (VOLY), i.e. the value of a YOLL needed for changes in life expectancy. This issue would be straightforward if the government provided guidelines with official values (as is the case in the USA for VPF). Unfortunately there are currently are no such guidelines for the EU. However, a report by OECD [2011] is increasingly used as reference. It is based on a meta-analysis by Lindhjem et al [2011] of all available stated preference surveys [9] and recommends a base value VPF of $\$_{2005}$ 3.5 million for EU27, with an uncertainty range of 1.75 to 5.25 million; it also discusses VOLY but without recommending a value. Based on a critical review of the literature and the OECD report, the commission of the French government on evaluation of public projects [Quinet 2013] recommends a VPF of 3 million $€_{2010}$ and a VOLY of 115,000 $€_{2010}$, to be adjusted in future years in proportion to consumer price index and GDP/capita. This has become de facto the guideline for France [Quinet 2015]. In view of this situation we use the numbers of Quinet to obtain a VPF of 3.3 million $€_{2013}$ and a VOLY of 126,000 $€_{2013}$, after adjusting for inflation and GDP growth and some rounding.

For fatal cancers there has been some debate whether one should use more than VPF because this form of death is particularly dreaded. However, Lindhjem et al [2011] did not find any clear evidence for a cancer premium and so we use the same VPF for cancer deaths. For infant deaths, by contrast, a premium does seem appropriate because parents care so much about their children and values in the range of 1.5 to 2 * VPF have been recommended [OECD 2011, Section 5.2]. Therefore we multiply VPF by 1.75 to obtain 5.8 million $€_{2010}$ for infant mortality.

Values for morbidity endpoints in the EU have been reviewed by several studies, especially ExternE [205, 2008], Hunt and Ferguson [2010], and Hunt [2011]. The number of original studies is much smaller than for VPF, and for many endpoints there are no data at all. One of the difficulties lies in the enormous variability of severity and duration between morbidity episodes of different individuals. Chronic bronchitis, for instance, can range from extended periods of coughing (unpleasant but not incapacitating) to constant and permanent breathing problems so severe that normal activities such as work are no longer possible. The analysis proceeds on the assumption that the unit costs are consistent with the severity of the endpoints observed in the corresponding epidemiological studies.

[8] often designated by the unfortunate term "Value of Statistical Life" (VSL) which tends to evoke angry reactions among non-economists. It is not the value of life, whatever that may be, but the willingness-to-pay to avoid an anonymous premature death.

[9] The alternative valuation method of wage-risk studies (favored in the US) tends to yield somewhat higher VPF numbers.

When no monetary values are available we see only one approach: use DALY [Salomon et al 2012] or QALY [see e.g. Sullivan and Ghushchyan 2006] weights if available, assuming a monetary value for the respective DALY or QALY. There are numerous debates about the extent to which DALY or QALY are compatible with monetary valuation, and there is no consensus. Here we use only DALY data. In the spirit of the development of these indices we find it most reasonable to set the value of a DALY equal to VOLY.

For neurotoxic effects we take as proxy a value of $\$_{2008}$ 18,000 per IQ point lost, following Spadaro and Rabl [2008a] who reviewed studies of the relation between IQ and lifetime earnings in the USA. There are no analogous original studies in the EU where IQ is not routinely measured; the value used by ExternE [2008] is based on Spadaro and Rabl [2008a]. We have found only one more recent value for an IQ point, $\$_{2009}$ 17,815 by Gould [2009]; it is also based on a review of the original studies and essentially the same value. Converting at a PPP adjusted exchange rate of 0.80 €/$ and adjusting for inflation this yields 16,300 $€_{2013}$ per IQ point.

One of the endpoints for arsenic is chronic bronchitis for which ExternE [2008] uses 200,000 $€_{2008}$ per case; that is based on two contingent valuation studies in the US as well as a valuation using QALY weights [Desaigues et al. 2007]. For non-fatal cancers we use INC [2007]. The numbers in this study imply that on average the treatment cost is 38,879 $€_{2007}$ and the productivity loss 60,432 $€_{2007}$. Adjusting for inflation we obtain the total market cost as 115,000 $€_{2013}$. Following ExternE [2008] we multiply by 1.5 to account for pain and suffering to obtain the unit cost of non-fatal cancers as 173,000 $€_{2013}$.

There are three endpoints for which we have found no explicit valuation and so we use DALY data. DALY weights are per year of the respective condition; in addition one has to estimate the duration of the condition, which is also problem. For fractures and childhood anemia we use the DALY weights of Salomon et al [2012], combined with our estimates of the duration. For fractures due to osteoporosis we take 19,500 € for treatment [Mutuelle Saint Martin 2012] and 1.17 DALY/case (0.308 first year + 12 years *0.072). For childhood anemia we take 0.058 DALY/yr for a duration of 70 yr. For diabetes we use the estimate of a total of 2.2 DALY/case by Huijbregts et al [2005].

Table 2. The unit costs P for health endpoints assumed in this paper.

End point	**This paper, $€_{2013}$**
VOLY (value of a life year), €/YOLL	126,000
VPF (value of prevented fatality)	3,300,000
Infant death = VPF * 1.75	5,800,000
Fatal cancer = VPF	3,300,000
Non-fatal cancer	173,000
Chronic bronchitis (CB)	226,000
Value of IQ point	16,300
Value of a DALY = VOLY	126,000
Diabetes [a]	277,000
Fracture due to osteoporosis [b]	167,000
Childhood anemia [c]	512,000

[a] 2.2 DALY/case [Huijbregts et al 2005]
[b] 19,500 € for treatment and 1.17 DALY/case

[c] 70 yr * 0.058 DALY/yr = 4.06 DALY/case

If there is a lag of n years between exposure and impact, we multiply the costs by a factor $(1 + r_{dis})^{-n}$ where r_{dis} is the social discount rate (for which we take 4% [10]). Lags are very different for different pollutants and different impacts. They are difficult to measure and there are few good data, as highlighted for instance in Howard [2013] (whose numbers would in any case not be suitable because here we need averages rather than lower limits). Most cancers develop slowly over many years. For fatal cancers typical lags are in the range of 10 to 30 yr, and here we assume a lag of 20 yr; that reduces their cost by a discount factor of 0.46. For general mortality, chronic bronchitis, diabetes, osteoporosis and anemia we assume a lag of 10 yr and a discount factor of 0.68. We assume that discounting is negligible for neurotoxic impacts because most of the damage occurs during the very first years of life and the assumed cost per IQ point has been calculated as discounted total loss of lifetime earnings at the time of birth. The total cost is the sum over all endpoints.

2.5. Accounting for thresholds

For cancers due to genotoxic carcinogens we follow the standard assumption that the ERF is linear without threshold at low doses. We also assume that there is no threshold for the impacts of Pb, a view that seems generally accepted in view of the fact that the ERF for neurotoxic effects has been measured down to very low exposures and found to be without threshold, and probably even above a straight line.

To find the threshold for all other endpoints, we use guideline values for maximum safe intake that have been established by organizations such as WHO or EPA for the protection of human health. However, we emphasize that such values have been established to ensure that nobody is exposed to significant risk, and they are not necessarily real thresholds. Even if the ERF were a hockey stick, the threshold would be difficult to determine because of all the uncertainties, especially for thresholds based on toxicology and the extrapolation from animals to humans. The hockey stick with maximum safe intake as threshold is an approximation but it is difficult to do better in view of the available information.

To account for a threshold it may seem natural to multiply the no-threshold result by the fraction of the population whose current exposure is above threshold. However, even with the assumption of a hockey stick, the fraction of the population above threshold is not what matters; rather, one has to determine the fraction of the total incremental exposure that is above threshold. That requires an analysis of the distribution of incremental exposures, as shown by Spadaro and Rabl [2008a] who carried out such a calculation for the damage cost of Hg. The fraction f_{thr} of the total incremental exposure that is above threshold is significantly larger than the fraction of the population above threshold. In the case of Hg we follow Spadaro and Rabl who take a threshold = 6.7E-03 mg_{Hg}/kg_{body}/day [EPA 2001] and find that f_{thr} is 0.44, about 5.5 times larger than the fraction of the population. The following lines explain the calculation of f_{thr}.

Without a threshold the total impact rate due to a total collective exposure E is

[10] See Table 9.1 of Rabl, Spadaro and Holland (2014) which summarizes a review of social discount rates. There has been a tendency to reduce the discount rates used by governments; now 6% by EU, 4% by France. In the USA the recommendation is to use both 3% and 7%.

$$I = s_{ERF}\, E \tag{8}$$

The collective exposure E is the sum of the individual exposures e_i over the entire population. If there is a threshold e_{thr} the total impact rate is

$$I_{thr} = s_{ERF} \sum_{i=p_{thr}}^{p} (e_i - e_{thr}) = s_{ERF} \sum_{i=p_{thr}}^{p} e_i - s_{ERF}\,(p - p_{thr})\, e_{thr} \tag{9}$$

where the individual exposures e_i are in increasing order and p_{thr}/p is the fraction of the affected population that is above threshold. If an incremental emission rate ΔQ increases the exposures by Δe_i the impact rate increases by

$$\Delta I_{thr} = s_{ERF} \sum_{i=p_{thr}}^{p} \Delta e_i \tag{10}$$

Let us assume, like Spadaro and Rabl, that the distribution of the incremental exposures Δe_i due to atmospheric emissions is like that of the e_i

$$\Delta e_i = e_i \;\; \Delta E/E \quad . \tag{11}$$

Whereas that assumption is realistic for Hg because most Hg is first emitted to the air (natural emissions being mostly volcanic), the distributions of Δe_i and e_i can be different for pollutants such as As because part of the total exposure comes from geological As that enters directly into the food chain. Unfortunately no information is available on the distribution of the Δe_i, and so we use Eq.11 with $\Delta E = IF\, \Delta Q$ to obtain the impact rate increase

$$\Delta I_{thr} = s_{ERF}\, IF\, \Delta Q \sum_{i=p_{thr}}^{p} e_i/E \quad \text{where } E = \sum_{i=0}^{p} e_i \quad . \tag{12}$$

As with the passage from Eq.3 to Eq.6 we thus obtain the damage cost D_{thr} for the threshold case

$$D_{thr} = P\, s_{ERF}\, IF\, f_{thr} \tag{13}$$

where

$$f_{thr} = \sum_{i=p_{thr}}^{p} e_i / \sum_{i=0}^{p} e_i \tag{14}$$

is the fraction of the incremental collective exposure that is above threshold.

For As, Cd and Cr we estimate the fraction f_{thr} on the basis of a recent survey of exposures in France where urinary concentrations of the toxic metals have been measured for a representative sample of the population [InVS 2011]. To relate the urinary concentrations to intake, we use conversion factors that we have found in the literature. The exposure distributions are very close to lognormal and the authors indicate the geometric mean μ_g. Therefore we choose the geometric standard deviation σ_g such that the resulting distribution is a good match for the data; σ_g turns out to be close to 2. Knowing the probability distribution it is straightforward to evaluate f_{thr} of Eq.14.

3. Uncertainty

The calculation of damage costs involves many assumptions and model parameters that are more or less uncertain. A rigorous uncertainty analysis would involve a detailed examination of each element of the calculation to estimate its probability distribution. Then the probability distribution of the result should be determined by a Monte Carlo calculation. That would be a major undertaking, way beyond the scope of this paper. But as a start we list in Table 3 the main elements of the impact pathway analysis with a qualitative indication of their uncertainties.

In particular the calculation of the intake fraction from ingestion is so complex that no Monte Carlo analysis has been published as far as we have been able to ascertain. The closest to an uncertainty assessment of intake fractions that we have been able to find are Spadaro and Rabl [2004] and Huijbregts et al [2004]. Spadaro and Rabl compare their results (which are based on the model of EPA [1998]) with the CalTox model [McKone and Enoch 2002] and find agreement within a factor of about 2 for As, Cr and Ni, and within a factor of 20 for Cd and Pb. Spadaro and Rabl also carry out a sensitivity analysis by varying the most important input parameters over a wide range (threefold for most): the corresponding change of the intake fraction is a factor of at most two. Huijbregts et al compare intake fractions for 367 substances as calculated by CalTox with those calculated by USES-LCA; they find that on average the disagreement between CalTox and USES-LCA can be characterized as a geometric standard deviation of about six to eight (the square root of their uncertainty factors which are for 95% confidence intervals).

Here we use, as a simple alternative to a detailed Monte Carlo calculation, the approach of Spadaro and Rabl [2008b] and Rabl, Spadaro and Holland [2014] who have shown that the uncertainty of the damage cost for an endpoint can be estimated in terms of lognormal distributions and geometric standard deviations. The lognormal distribution is appropriate because the calculation essentially a product of factors. In practice the distribution of a product is approximately lognormal even if the number of factors is small, provided the distributions of the most uncertain factors are themselves not too far from lognormal. Spadaro and Rabl [2008b] have found that to be the case for damage costs.

This finding has a simple interpretation: if the damage cost has a lognormal distribution with a geometric mean μ_g and a geometric standard deviation σ_g, the probability is approximately 68% for the true cost to be in the interval $[\mu_g/\sigma_g, \mu_g\, \sigma_g]$ and 95% for it to be in the interval $[\mu_g/\sigma_g^2, \mu_g\, \sigma_g^2]$, in other words

$$\text{the 68\% CI is } [\mu_g/\sigma_g, \mu_g\, \sigma_g] \quad \text{and} \quad \text{the 95\% CI is } [\mu_g/\sigma_g^2, \mu_g\, \sigma_g^2] \quad . \tag{15}$$

Table 3. The main elements of the impact pathway analysis (IPA) and main sources of uncertainty. Not explicitly listed is the role of choices by the analyst when the available information is not sufficient (being incomplete, only indirectly relevant, ambiguous, contradictory or too difficult to find). The magnitude of the uncertainties can be very different from case to case.

Step of IPA	**Element**	**Main Uncertainties**
Emission	for calculation per kg of pollutant for calculation per unit product (kWh, tonne waste etc)	None. emission per unit product can be quite uncertain.
Dispersion (calculation of exposure or IF)	Exposure from inhalation Exposure from ingestion	Modeling of atmospheric dispersion, wet and dry deposition, and for reactive pollutants their chemical transformations. Modeling the pathways of the pollutants into soil, water and the food chain. Delays between emission and exposures.
Impact (ERF)	RR Other factors needed for ERF Extrapolation to lower exposures	Confidence intervals as reported. Possible biases of the epidemiological studies because of their design. Background rates Exposure conversion factors Extrapolation from study population to population of concern. Form of ERF (linearity, threshold).
Cost (Monetary valuation)	Studies of willingness-to-pay (WTP)	Confidence intervals as reported. Variability between studies
	Linking impact to cost estimates	Relation between endpoint in epidemiological study and endpoint for which there are cost data.
	Discounting of impacts that occur in the future	Time of onset, discount rate, severity of impacts (note medical progress).
	Benefit transfer	Extrapolation from population of WTP study to population of concern.

The geometric standard deviation σ_g of the product $z = x_1\, x_2\, x_3 \ldots x_n$ of uncorrelated factors x_i is given by

$$[\ln(\sigma_g)]^2 = [\ln(\sigma_{g,1})]^2 + [\ln(\sigma_{g,2})]^2 + \ldots + [\ln(\sigma_{g,n})]^2 \quad . \tag{16}$$

where $\sigma_{g,i}$ is the geometric standard deviation of x_i. That equation is exact. Thus it suffices to estimate the $\sigma_{g,i}$ for each of the factors of the damage cost calculation. For factors whose confidence intervals are not too asymmetric, an equivalent geometric standard deviation can be estimated by the equation

$$\sigma_g = \sqrt{\frac{\mu+\sigma}{\mu-\sigma}} \qquad (17)$$

where μ is the ordinary mean and σ the ordinary standard deviation. That is the case in particular for the RR of most epidemiological studies, and so their σ_g can be estimated as the fourth root of the ratio of the upper and lower bounds of the 95% confidence interval.

Thus it suffices to estimate the geometric standard deviations for each of the key factors of the damage cost calculation, namely intake fraction, exposure-response function (ERF), and monetary valuation. For the ERF we consider not only the relative risk RR of a health effect but also the corresponding additional factors such as the relation between biomarker and intake. Even though the final result is the sum over endpoints, one can estimate σ_g by looking at the endpoint with the highest damage cost, as shown by the examples in Section 11.4 of Rabl, Spadaro and Holland [2014].

Compared to a Monte Carlo analysis of uncertainties, our approach has the advantage of being transparent: the reader can readily modify the $\sigma_{g,i}$ to see the effect on the resulting σ_g. A Monte Carlo result is a just a number and it is difficult to show how it would change with different assumptions inside the black box. Even though our approach is approximate, it is sufficient for practical purposes because the utilization of an environmental cost-benefit analysis is not sensitive to the precise value of the uncertainty: for example the choice of a decision maker is unlikely to be different whether σ_g is 3.5 or 4.5.

For the intake fraction we argue that the results of Huijbregts et al are far too pessimistic. The environmental pathway modeling of atmospheric emissions has to begin with the dispersion in the atmosphere, and that step determines the inhalation intake fraction. Comparing the inhalation intake fraction results between CalTox and USES-LCA, Huijbregts et al find that the disagreement corresponds to geometric standard deviations larger than 30. As explained in the following paragraph, such enormous disagreement stems from unrealistic modeling of atmospheric dispersion by USES-LCA.

Long term inhalation exposure due to primary pollutants (including metals) can be calculated with geometric standard deviations of 2 or better, as shown by numerous validation studies, including the Monte Carlo analysis of Spadaro and Rabl [2008b] and comparisons between calculated and measured concentrations [e.g. Rabl, Spadaro, Holland 2014]. Furthermore the ratio of ingestion dose and inhalation dose of metals has generally been observed to be in the range of several tens to several hundreds (see e.g. the reports by WHO and ATSDR [11]). Thus the ratios of ingestion over inhalation intake of Spadaro and Rabl [2004], about 50 to 80 in Table 1, are very plausible. Exposure distributions are approximately lognormal, and the ratios of lognormal quantities are also lognormal. Taking simple numbers for the sake of illustration, if 68% of the ratios ingestion/inhalation are between 30 and 300, σ_g for that ratio is the square root of 300/30 = 3.16..., and if 95% of the ratios ingestion/inhalation are between 30 and 300, σ_g for that ratio is the fourth root of 300/30 = 1.778.... That suggests that the ratio ingestion/inhalation for typical conditions can be estimated within a factor of about 2 to 3 even without calculation. We also note that the sensitivity analysis in Table A5 of Spadaro

[11] Of course the ratios implied by measured data have to be interpreted with caution because they may not correspond to steady state conditions and part of the ingestion dose may be due to metals of natural origin in soil or ground water.

and Rabl [2004] indicates an uncertainty range of about 0.4 to 2, very much smaller than implied by Huijbregts et al. If, for the sake of illustration, σ_g is 2 for the inhalation dose and 2 (or alternatively 2.5) for the ratio ingestion/inhalation, Eq.16 implies that σ_g for ingestion is 2.7 (or alternatively 3.2). In view of these considerations we set σ_g for intake equal to 3.

Table 4 shows our choices for the $\sigma_{g,i}$ of the key factors of the damage cost calculation. For monetary valuation we take $\sigma_{g,i}$ to be 2, based on Spadaro and Rabl [2008b]. The last column of this table shows the contribution of the $[\ln(\sigma_{g,i})]^2$ to the total of Eq.16. Because of the quadratic combination of terms only the largest $\sigma_{g,i}$ make a significant contribution. The resulting geometric standard deviation σ_g of the damage cost is 4.1, which we round off to 4. We emphasize that these are typical estimates; for specific endpoints of specific metals the uncertainty can be somewhat different as the reader can test by changing the $\sigma_{g,i}$ in Eq.16. Additional uncertainty due to thresholds can be estimated by comparing the results with and without threshold in Table 5.

Table 4. Typical $\sigma_{g,i}$ of the key factors of the damage cost calculation.

	$\sigma_{g,i}$	$ln(\sigma_{g,i})^2$
Intake fraction IF	3	*1.21*
Relative risk RR	1.5	*0.16*
Other factors for ERF	1.5	*0.16*
Monetary valuation	2	*0.48*
Total σ_g	**4.1**	***2.02***

In this kind of work there is always the nagging question: "are there additional significant impacts that we do not yet know about?" This very paper is a warning about such a possibility, because our damage costs are so much higher than previous estimates for As, Cd and Pb by ExternE [2008]. However, those previous estimates were quick and dirty jobs using only readily available ERFs, namely IQ loss due to Pb, and cancers based on unit risks of EPA (generally out of date). Only in recent years have enough epidemiological studies been carried out to yield ERFs for the most costly endpoints of these metals. In view of the scope of the studies now available we believe that the order of magnitude of our estimates is correct, but we cannot offer any guarantee.

Likewise one may wonder about ecosystem impacts, so far not addressed. These metals are natural ingredients in many soils, albeit in minute quantities. Some, especially Hg, enter the atmosphere via volcanic eruptions (about a third of world wide Hg emissions are of natural origin). In some regions As from soil enters the water supply in much higher quantities than from anthroprogenic sources, without any noticeable impact on ecosystems. Plants are quite insensitive to As, Cd, Hg and Pb, so the only significant impact of added exposure from atmospheric emissions could be on animals. Ecosystem impacts are valued at the level of species or entire ecosystems, whereas human health impacts are valued at the level of individuals, with very high monetary values. To appreciate what this implies, consider that the RR for age-specific mortality of the most exposed individuals in the epidemiological studies (due to much higher pollution levels of the past) are around 1.5 which shortens LE by about 5%. For humans that is a dramatic loss, reflected in very high valuation. Most of that mortality is due to cancers and heart disease and occurs well after reproduction. How could an equivalent effect on animals have a major impact on ecosystems?

4. Results and Conclusions

Our damage cost estimates are summarized in Table 5. They include discounting at a discount rate of 4%. The choice of the threshold, if any, is indicated, together with the fraction f_{thr} of the incremental exposure above threshold. For comparison we also show the damage costs of ExternE [2008]; they are much lower because only cancers were taken into account for As and Cd, and only IQ loss for Hg and Pb.

For the derivation of the numbers in Table 5, see our companion papers in this series, Part 2 for Hg and Pb (in this issue of Risk Analysis) and Part 3 for As and Cd (not yet published but available at http://www.arirabl.org/software/). The Excel file with the calculations is available as Supplemental File; it can also be downloaded from http://www.arirabl.org/software/).

Since these metals (with the exception of much of the Hg) condense onto particles in the air and $PM_{2.5}$ causes severe health impacts, the reader might wonder if the ERFs for $PM_{2.5}$ should also be taken into account. That would raise questions of double counting and questions about the role of the composition of $PM_{2.5}$ for those ERFs. However, the damage cost per kg of $PM_{2.5}$ emitted by industrial installations is less than 100 €/kg, and even if one were to simply add such a contribution, it would be negligible.

Table 5. Summary of damage costs contributions for each of the metals, in €$_{2013}$/kg, for industrial emissions in the EU.
Cases are without f_{thr}, with units YOLL for non-cancer mortality and IQ points for IQ loss.
f_{thr} *= fraction of incremental exposure above threshold.*

a) Arsenic

Threshold = 3.0E-04 mg_{As}/kg_{body}/day *of EPA, applied to all endpoints.* f_{thr} *= 0.80.*
ExternE [2008] had estimated 530 €/kg$_{As}$.

Endpoint	**Cases/kg$_{As}$**	**Undiscounted, no threshold**	**Lag [yr]**	**Discount factor**	**Discounted, no threshold**	**Discounted, with threshold**
Non-cancer mortality	9.95E-03	3,909	10	0.68	2,641	2,120
Cancer deaths	5.96E-04	1,966	20	0.46	897	720
Non-fatal cancers	6.54E-04	113	20	0.46	52	41
Chronic bronchitis	1.58E-03	357	10	0.68	241	194
IQ loss	4.93E-02	803	0	1.00	803	645
Infant deaths	9.88E-06	57	10	0.68	39	31
Diabetes	1.30E-02	3,617	10	0.68	2,443	1,962
Total cost, €$_{2013}$/kg$_{As}$		**10,821**			**7,115**	**5,713**

b) Cadmium.

Threshold = 0.1 μg/kg_{body}/day [ATSDR 2012]*, applied to all endpoints.* f_{thr} *= 0.98.*
ExternE [2008] had estimated 84 €/kg_{Cd}.

Endpoint	Cases/kg_{Cd}	Undiscounted, no threshold	Lag [yr]	Discount factor	Discounted, no threshold	Discounted, with threshold
Mortality	1.633	205,781	10	0.68	139,018	136,752
Non-fatal cancers	0.005	937	20	0.46	428	421
Fractures	0.016	2,703	10	0.68	1,826	1,796
Total €$_{2013}$/kg_{Cd}		**209,421**			**141,272**	**138,969**

c) Mercury.

Threshold = 6.7E-03 mg_{Hg}/kg_{body}/day [EPA 2001] and f_{thr} = 0.44.
ExternE [2008] had estimated 8,000 €/kg_{Hg}.

Endpoint	Cases/kg_{Hg}	Undiscounted, no threshold	Lag [yr]	Discount factor	Discounted, no threshold	Discounted, with threshold
Mortality	0.56	70,085	10	0.68	47,347	20,833
IQ loss	1.36	4,782	0	1	4,782	2,104
Total €$_{2013}$/kg_{Hg}		**74,867**			**52,129**	**22,937**

d) Lead.

Threshold = 0 and f_{thr} = 1 for all endpoints.
ExternE [2008] had estimated 278 €/kg_{Pb}.

Endpoint	Cases/kg_{Pb}	Undiscounted, no threshold	Lag [yr]	Discount factor	Discounted, no threshold
Mortality	0.29	36,796	10	0.68	24,858
IQ loss	0.27	4,435	0	1	4,435
Anemia	1.44E-04	74	10	0.68	50
Total €$_{2013}$/kg		**41,305**			**29,343**

It is interesting to look at the implications of these results for combustion of coal and incineration of waste. Table 6 shows the damage costs of waste incinerators due to atmospheric emission of toxic metals, together with CO_2 and the classical air pollutants. The damage costs of PM_{10}, SO_2 and NO_2, in Tables 6 and 7, are based on ExternE [2008] but updated with the unit costs in Table 2.

The emissions can vary from one installation to another and getting representative measured data is not easy. Therefore we show both the emission limits in the EU and the actual emissions in France. Since the emission limits are formulated in terms of flue gas concentrations, we have translated them to g/t_{waste} by assuming 5150 Nm^3/t_{waste}. The data for incinerators in France are based on CITEPA [2013], the European Pollutant Release and Transfer Register (E-PRTR) and discussions with Olivier Guichardaz of Dechets-Infos. The toxic metals make a very significant contribution, especially Cd, Hg and Pb. To put the numbers in perspective, note that the private cost of waste incineration is on the order of

100 €/t_{waste}: the damage costs are still very significant, even after major reductions compared to incinerators of the past.

Table 6. Damage costs due to atmospheric emissions by waste incinerators.

		Limit values EC [2000]		Actual emissions, France 2011	
Pollutant	**€$_{2013}$/kg**	**g/t waste**	**€$_{2013}$/t waste**	**g/t waste**	**€$_{2013}$/t waste**
CO_2	0.040	863867 [a]	34.55	863867 [a]	34.55
PM_{10}	39.00	51.5	2.01	7.3	0.29
SO_2	17.50	257.5	4.51	54.3	0.95
NO_2	16.30	1030	16.79	600.0	9.78
As	5713	0.072	0.41	0.013	0.08
Cd	138969	0.209	29.06	0.007	0.93
Hg	22937	0.258	5.91	0.053	1.22
Pb	29343	0.567	16.62	0.093	2.74
Total			**109.86**		**50.54**
Toxic metals			**52.00**		**4.96**

[a] actual emissions because no limit value for CO_2

Both coal and oil contain toxic metals such as As, Cd, Pb and Hg, some of which escapes through the smoke stacks of power plants. The amounts of these trace metals can vary greatly with the origin and type of oil or coal. In Table 7 we show emissions data for hard coal condensing power plants in the EU27 according to ExternE [2008], together with our damage costs. The largest contributions are due to Hg and Pb, but even those are small compared to the damage costs of the classical air pollutants and greenhouse gases, and compared to the private cost of electricity, averaging around 20 €cent/kWh for households and 12 €cent/kWh for industry in the EU, but quite variable from country to country.

However, what matters for policy applications is not the absolute magnitude but the comparison of the damage cost to the cost of pollution abatement (both upstream and end of pipe) for each pollution source. New cost-benefit studies for the abatement of toxic metal emissions are advisable, with particular attention to older more polluting plants that may still be operating. We also emphasize that even though the uncertainties are large, it is not the uncertainties themselves that matter but their effect on policy choices. As shown by Rabl, Spadaro and van der Zwaan [2005], despite such uncertainties the results are very useful for environmental policy because they can help avoid costly mistakes.

Table 7. Emission (to air) and damage costs for coal fired power plants. Emissions data of ExternE [2008].

Pollutant	€$_{2013}$/kg	kg/kWh	€cent$_{2013}$/kWh
CO_2	0.040	7.30E-01	2.920
PM_{10}	39.00	2.74E-05	0.107
SO_2	17.50	5.51E-04	0.965
NO_2	16.30	5.54E-04	0.903
As	5713	7.98E-09	0.005
Cd	138969	3.92E-10	0.005
Hg	22937	2.54E-08	0.058
Pb	29343	3.43E-08	0.101
Total			**5.06**
Toxic metals			**0.17**

The methodology demonstrated in this paper should likewise be used for any other pollutant that might entail significant health impacts. In particular we have found that new epidemiological findings yield impacts that are far larger than what had been suspected. This highlights once again [12] the importance of periodically performing or revising studies of this kind, with an eye open to possible surprises.

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Abbreviations and symbols

D = damage cost, €/kg
DALY = disability adjusted life year
E = collective exposure, as intake in mg/yr
e = individual exposure, as intake in mg/yr
EPA = Environmental Protection Agency of USA
ERF = exposure-response function
f_{thr} = fraction of collective incremental exposure that is above threshold
I = impact rate, cases/yr
IF = intake fraction, mg/kg
IPA = impact pathway analysis
LE = life expectancy
NHANES = National Health and Nutrition Examination Survey
OR = odds ratio
P = unit cost ("price") of end point, €/case
PM = particulate matter (subscript indicates largest diameter, in μm)
PPP = purchasing power parity

[12] A similar surprise happened when externality studies started around 1990 and found health impacts of air pollution far larger than expected.

Q = emission rate of pollutant, kg/yr
QALY = quality adjusted life year
RfC (RfD) = reference concentration (dose) = maximum ambient concentration (dose) for lifetime exposure that is not likely to cause harmful effects
RR = relative risk
s_{ERF} = slope of ERF, cases/mg
VOLY = value of a life year
VPF = value of prevented fatality = VSL = "value of statistical life"
WHO = World Health Organization
WTP = willingness-to-pay
YOLL = years of life lost

μ_g =geometric mean
σ_g = geometric standard deviation

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